Collectively, these studies identify a cell-intrinsic role for IGF signaling in zebrafish primordial germ cell development.

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Program/Abstract # 276
Magellan functions during oogenesis to establish the animal–vegetal axis of the zebrafish egg
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In vertebrates, the genetic basis for the establishment of oocyte polarity is not well understood. We isolated a mutation called *magellan* (*mgn*) that affects animal–vegetal (AV) polarity of the zebrafish egg. In wild-type eggs, cytoplasm accumulates in the blastodisc at the animal pole. In contrast, cytoplasm in eggs from *mgn* mutants surrounds the yolk, indicating a defect in AV polarity. Additionally, a single micropyle, through which sperm enters the egg, marks the animal pole in wild-type eggs, while eggs from *mgn* mutants have variable numbers of micropyles, further indicating a defect in AV polarity. In zebrafish, the position of the Balbiani body, a highly conserved structure that includes ER, mitochondria and germ plasm mRNAs, predicts the AV axis during oogenesis. The Balbiani body has been found in all organisms examined from *Drosophila* to mammals, but its function has thus far remained unclear. In zebrafish, the Balbiani body forms on the future vegetal side of the oocyte, thus marking the AV axis. In wild-type oocytes, the Balbiani body forms during early stage I, localizes mRNAs to the vegetal pole and disassembles by stage II. Analysis of *mgn* mutant ovaries revealed an abnormal persistence of the Balbiani body in stage II and III oocytes. Furthermore, mRNAs that normally localize to the Balbiani body during stage I are found in the persistent Balbiani body of stage II and III mutant oocytes. Our data suggest a critical role for regulation of the Balbiani body by *mgn* in the establishment and/or maintenance vertebrate oocyte polarity. We will present our progress in determining the molecular nature of the *mgn* gene.

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Program/Abstract # 277
Ovarian development in mice requires GATA4/FOG2 transcriptional complex
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We have demonstrated previously that mammalian sexual differentiation requires both GATA4 and FOG2 transcription regulators to assemble the functioning testis. We have now determined that the sexual development of female mice is profoundly affected by the loss of GATA4–FOG2 interaction. We report here that the GATA4/FOG2 complex is required to activate several genes with an established role in ovarian development (e.g., *Wnt4*, *follistatin* (*Fst*) and *Foxl2*) as well as to turn on the rest of the sexually dimorphic ovarian program. We have also identified the *Dkk1* gene, encoding a secreted inhibitor of canonical β-catenin signaling as a target of GATA4/FOG2 repression in the developing ovary. The tissue-specific ablation of the β-catenin gene in the gonads disrupts female development while in the $Gata4^{ki/ki}/Dkk1^{-/-}$ or $Fog2^{-/-}/Dkk1^{-/-}$ embryos the normal ovarian gene expression pattern is partially restored. Control of ovarian development by the GATA4/FOG2 complex presents a novel insight into the crosstalk of transcriptional regulation and extracellular signaling in ovarian development.

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Program/Abstract # 278
CDC14A and CDC14B regulate meiotic progression in mouse oocytes
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Meiosis generates haploids from diploid precursor cells and is essential for all sexually reproducing organisms. Errors in the first meiotic division (MI) are linked to chromosomal nondisjunctions and occur at a higher frequency in females compared to males. Little is known, however, about how this unique segregation is regulated. CDC14 is a highly conserved, dual specificity phosphatase that is required for centrosome duplication, mitotic exit and cytokinesis in somatic cells. In budding yeast, *CDC14* mutants fail to properly exit from MI, thus generating aneuploid spores (gametes) that are inviable. *Cdc14′s* meiotic function in higher eukaryotes is not known. In this study we used the mouse oocyte to study the roles CDC14A and B play during meiotic progression. We found by immunofluorescence that these proteins are present in different subcellular locations in the oocyte except at AI when they co-localize on the central region of the meiotic spindle. Knockdown of *Cdc14b* by RNA interference causes premature germinal vesicle breakdown (GVBD) whereas over-expression of *Cdc14b* delays GVBD, suggesting that CDC14B is a regulator of maintaining the prophase I arrest in meiotically incompetent oocytes. Microinjection of an antibody that disrupts the function of CDC14A into oocytes results in chromosome misalignment on the meiosis II (MII) spindle and alters the kinetics of the MI–MII transition. These data suggest that CDC14B acts prior to CDC14A and indicate that CDC14A and B have distinct functions during female meiosis that are both critical for the development of a healthy egg.

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Program/Abstract # 279
Hsp90a regulate meiotic G2/M transition in mouse oocyte
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Heat shock proteins (Hsps) are molecular chaperones involved in the folding and the activity of numerous proteins. Although Hsps were reported to be expressed in the oocytes and therefore could serve as important maternal factors, there was no comprehensive picture of the oocyte specific Hsp “chaperome”. Using RTqPCR, we analysed Hsp25, 60, 70.1 and 70.3, the two isoforms Hsp90a and Hsp90b and Hsp105. We discovered that Hsp90a was the most abundant chaperone in murine oocytes and we found that this high level of expression required the transcription factor, HSF1. To determine the function of HSP90a, we used a specific inhibitor, 17-allylaminogeldanamycin (17-AAG) which blocks the ATPase activity of Hsp90a and